Perforative Peritonitis assessment of severity using modified APACHE II Score

Dissertation Submitted
for the Degree of

MASTER OF SURGERY

Branch I

(GENERAL SURGERY)

THE TAMIL NADU
Dr. M.G.R. Medical University
CHENNAI

SEPTEMBER 2006

Coimbatore Medical College

Coimbatore - 641 014

Certificate

Certified that this is the bonafide dissertation done by

Dr. K. KARTHIKEYAN

and submitted in partial fulfilment of the requirements for the

Degree of M.S., General Surgery, Branch I of

The Tamilnadu Dr. M.G.R. Medical University, Chennai

Date : Unit Chief

Date : Professor & Head
Department of Surgery

Date : Dean
Coimbatore Medical College
Coimbatore - 641 014

Declaration

I solemnly declare that the dissertation titled "**Perforative Peritonitis assessment of severity using modified APACHE II Score**" was done by me during the period January 2005 – December 2005 under the guidance and supervision of Professor **Dr. K.P. Arunkumar, M.S.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the regulation for the award of MS Degree in General Surgery (Branch I).

Place **Dr. K. KARTHIKEYAN**

DATE :

ACKNOWLEDGEMENT

At the outset I thank **Dr. T. P. KALANITI M.D.,** our beloved Dean, Coimbatore medical college, for having permitted me to use the hospital material for study.

I express my deep sense of gratitude to Prof. **Dr. K.P. ARUNKUMAR M.S.,** Head of the Department and Unit Chief for his guidance in preparing this dissertation.

I thank professors **Dr. PERUMAL RAJAN, M.S., Dr. ESWARAN, M.S., Dr. RAMAMOORTHY, M.S.,** for their suggestions and kind help in doing my study.

I record my thanks to all the Assistant Professors for their valuable guidance and encouragement in my study.

Last but not the least I thank all the patients for their whole hearted co-operation without which the work would not have been possible.

CONTENTS

INTRODUCTION

Acute generalized peritonitis from Gastrointestinal hollow viscus perforation is a potentially life threatening condition. It is a common surgical emergency in many general surgical units in the developing countries and it is often associated with high morbidity and mortality.[1,2] Grading the severity of acute peritonitis has assisted in no small way in decision making and has improved therapy in the management of severely ill patients[3], Empirically based risk assessment for important clinical events has been extremely useful in evaluating new therapies, in monitoring resources for effective use and improving quality of care[4,5,6]. The introduction of injury severity scale by Baker's et al[7] in 1974 and abbreviated injury scale[8] in 1981 successfully opened avenues from further development of severity grading systems.

Many scoring systems have been designed and used successfully to grade the severity of acute peritonitis and abdominal sepsis.

The most widely used index APACHE (Acute physiology and chronic Health Evaluation) was developed from a mixed group of medical and surgical patients. It has been successfully used to assess critically ill general surgical patients and also been compared with other scoring systems with good results.[4,9,10,11,12]

AIM OF THE STUDY

Assess the severity of generalized peritonitis from hollow viscus perforation using modified APACHE II Score.

To study various types of perforative peritonitis as occurring in CMC hospital and their progression.

To find out the incidence of perforative peritonitis in relation to age group and sex of the patient.

To analyse the various symptoms and signs of the diseases from the onset of perforation.

Correlate morbidity and mortality patterns with the modified APACHE II Score and its significance on the outcome.

HISTORICAL DATA

The papyrus "Ebera" of Egypt mentioned the peritoneal cavity about 3500 years ago. However it was described first in 1730 A.D by James Douglas of Edinburgh. According to Lister the earliest case of acute perforation of a peptic ulcer producing peritonitis was recognized in 1070.

Credit of presenting the first duodenal ulcer perforation has gone to HAMBURGER in 1746. HEUSNER – pioneered the simple closure technique of perforated gastric ulcer in 1892.

The first repeated successful closure of perforated duodenal ulcer was by Dean in 1894. In 1929 Cellan Jones[13] developed the technique of using liver omental support in closure of perforation. ROSCOE GRAHAM in 1937 described the technique of closure of perforation with a free omental patch.

Imhofen et al in "Chirugury" 1987 [14] September 58 (9) discussed new therapeutic aspects in the treatment of peritonitis. According to it the use of solution applied for lavage having an alkaline pH, instead of neutral pH significantly improved the rate of survival.

Scoring systems was introduced by Baker's et al. Knaus et al first introduced APACHE II scores and used successfully in critically ill patients. Modified APACHE II Scores have been used in peritonitis to assess severity with good results.

REVIEW OF LITERATURE

A. Scoring systems and risk assessment Modified APACHE II Scores in generalized peritonitis.

B. Peritonitis

C. Surgical anatomy of peritoneal cavity

D. Surgical physiology

E. Pathophysiology

F. Bacteriology

G. Classification

H. Clinical Features and diagnosis

I. Differential Diagnosis

J. Investigation

K. Management

L. Complications

M. Prognosis

A. RISK ASSESSMENT USING SCORING SYSTEMS
MODIFIED APACHE II SCORES

Scoring systems give an objective method to assess the severity of disease and outcome.

Patient admitted in a intensive care form a heterogenous population.

They differ in many aspects including, age, pervious health status, reason for admission, severity of illness.

All factors influence the prognosis of the patient scoring systems have been developed to quantify this case mix.

Multivariate assessments provide a cumulative score made of collective contribution of various data which reflect the overall risk and therefore the outcome.[15]

Some of these are generic i.e. they can be used across a wide range of disease states and others refer to specific disease processes.

Some of the generic scoring systems are :

ASA – American society of Anaethesiology.[16]

APACHE – Acute physiology and chronic health evaluation

POSSUM – Physiological and operative severity score for the enumeration of mortality and morbidity.[9]

Disease specific scoring systems

Commonly used are cirrhosis of liver, pancreatitis, head injury, trauma and tumor prognosis.

Mannheims peritonitis index. [17]

Child's score, Paul Brousse Hospital's for cirrhosis classification.

Ranson's, Glasgow Imrie for pancreatitis.

Glasgow coma scale, Injury severity scale, revised trauma scale, for trauma and Head Injury.

APACHE II SCORES

This method has been the most validated and is now been widely accepted in intensive care setting.

Since generalized peritonitis is an acute surgical problem. APACHE SCORES are well correlated in assessing the severity of the disease and outcome.

The APACHE system gives a score, which is sum total of

1. Acute physiological score (APS)
2. Age Points
3. Chronic health points

In APACHE – II – APS is made of multiple variables. The weighting system is based on a scale of 0 (normal range) to 4 (high or low abnormal). The most deranged physiological value for each parameter on admission is used.

In our study a modified APS was used as given by Meakin et al.[12] Since arterial pH and PO_2 were not available in our hospital these parameters were scored zero. And the remaining parameters were included. Serum urea was included and was scored similar to serum creatinine (See table).

Age Points

Chronological age is an independent variable in its own right and for this reason points are assigned to the age in years as follows.
44 and below (0) 45-54 (2) 55-64 (3) 65-74 (5) 75 and above (6)

Chronic health points

As outcome is adversely influenced by previous history of severe organ or system disorders and immunodeficiency states points are allocated for these problems (See Table).

Modified APACHE II SCORES Meakin et al.,[12]

Physiological variables	+4	+3	+2	+1	0	+1	+2	+3	+4
Temp	>41	39-40.9		38.5-38.9	36-38.5	34-35	32-33.9	30-31.9	<29.9
Mean arterial BP	>160	139-159	110-129		70-109		50-69		<49
Heart Rate	>180	140-179	110-139		70-109		55-69	40-54	<39
Resp. Rate	>50	35-49		25-34	12-24	10-11	6-9		<5
Oxygenation PaO2 (mmHg) *	>500	350-499	200-349		<200				
Arterial Ph*	>7.7	7.5-7.59		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	<7.15
Serum Na+ mMol/l	>180	160-179	155-159	150-154	130-149		120-129	111-119	<110
Serum K+ mMol/1	>7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		<2.5
Serum Creat. Mg/100ml	>3.5	2-3.4	1.5-1.9		0.6-1.4		<0.6		
Haemtocrit %	>60		50-59.9	46-49.9	30-45.9		20-29.9		<20
WBCx1000 (total mm3)	>40		20-39.9	15-19.9	3-14.9		1-2.9		<1
Serum HCO3 Venous blood mMol	>52	41-51.9		32-40.9	22-31.9		18-21.9	15-17.9	<15
Serum Urea (mMol/l)	>15	9-14	5-9		1-4.9		<1		

*Arterial pH and PaO^2 were scored zero.

Age points for adults : < 44-0; 45-54 – 2; 55-64-3; 65-74 - 5, >75 - 6

Chronic ill – health evaluation (severe organ insufficiency) points; Presence of chronic illness in patients requiring the following:

1. Liver – biopsy proven cirrhosis, Portal hypertension – Upper GI bleed due to portal hypertension. Prior episodes of hepatic failure / encephalopathy/coma.

2. Cardiovascular : Newyork Heart Association Class IV

3. Respiratory : Chronic restrictive, obstructive or vascular disease reslting in severe excise restriction documented chronic hypoxia, hypertension > 40 mm Hg or Respiratory dependent.

4. Renal : Receiving chronic dialysis.

5. Immuno compromised – The patient has received theraphy that suppresses resistance to infection (eg) Immuno suppression, chemotherapy, radiation, steroids, diseases like leukemia, AIDS, lymphoma.

(a) for non – operative or emergency postoperative patients – 5; (b) for elective post operative patients – 2 points.

Mortality and Modified APACHE II

There is a clear cut inverse correlation between APACHE scores and survival. Higher score sharply increases mortality.

In our study we have used the modified APACHE II scores as suggested by Meakin et al in generalized peritonitis[1,18]. The results have been studied earlier successfully with good correlation.

B. PERITONITIS

Peritonitis[19] is defined as the inflammation of a portion or all of the parietal and visceral surfaces of the abdominal cavity. Secondary peritonitis may complicate any abdominal condition like trauma, infection obstruction or neoplasm.

A. Perforation is one that extends through the wall of the gastrointestinal tract, establishes communication between the lumen of the viscus and the surrounding peritoneal cavity and permits free flow of luminal contents into the peritoneal cavity, causing peritonitis.

C. SURGICAL ANATOMY OF PERITONEAL CAVITY

Development

The primitive coelom is divided into peritoneal cavity and pericardial cavity[20] by the septum transversum in the 4th week of intrauterine life. They communicate dorsally through the pleuro peritoneal canals. The peritoneal cavity in completely separated from pleural cavity by the diaphragm in the 7th week of intrauterine life. Initially the peritoneal cavity is separated into right and left halves by the gut and its dorsal and ventral mesenteries. Later, the lesser omentum and falciform ligament of liver persists while the whole of the ventral mesentery disappears. Two diverticula appear at the junction of foregut and midgut, the hepatic diverticulum grows into the ventral mesentery and pancreatic diverticulum into the dorsal mesentery. Spleen develops in the dorsal mesentery of stomach. With rotation of gut, the ventral mesogastrium into the right with the contained liver to become the lesser omentum. The dorsal meosgastrium shift to the left with the spleen. Fusion of left leaf of the dorsal mesogastrium with the parietal peritoneum forms the lienorenal ligament. The rest of the dorsal mesogastrium balloons out and grows down to fuse with the mesocolon after folding on itself posteriorly. The ventral mesogastrium forms the lesser omentum, enclosing liver in its layer and finally attached itself to the parietal layer as the falciform ligament, coronary ligament and triangular ligament. The rest of the coelom develops around the intestines as they are finally drawn into it. It covers rectum, bladder and in addition genital organs in the female.

Surgical Anatomy

The peritoneum is a thin serous membrane lining the walls of the abdominal and pelvic cavities and clothing the abdominal and pelvic viscera. It consists of parietal and visceral peritoneum.

The parietal peritoneum lines the walls of the abdominal and pelvic cavities. It is thicker than visceral peritoneum. It is innervated by somatic afferent nerves. It is quite sensitive to pain and accurately localised to the affected part.

Visceral peritoneum covers the viscera and mesentery. Sensory supply is through the autonomic nervous system. So it is poorly sensitive to pain, temperature, but responds to stretch and distention. Pain arising from this is vague and poorly localised.

The potential space between the parietal and visceral layers of peritoneum is called the peritoneal cavity. In male this is a closed cavity, but in the female there is a communication with the exterior through the uterine tubes, uterus and vagina.

Divisions of peritoneal cavity are

1. **Abdominal part / peritoneal cavity proper**
2. **Pelvic part**

The abdominal part is further divided into supracolic compartment and infracolic compartment by the transverse colon and transverse mesocolon

a. **Supracolic compartment** is divided in to spaces by liver and its ligaments and stomach. They are
    1. Right anterior (right sub phrenic)
    2. Right posterior (right Subhepatic or Morrison's pouch)
    3. Left anterior (left subphrenic)

4. Left posterior (left sub hepatic or lesser sac)

b. Infracolic compartment is divided into four regions by the ascending and descending colon, the root of the mesentery and the pelvic mesosigmoid.

1. Right lateral paracolic gutter
2. Right medial paracolic gutter
3. Left lateral paracolic gutter
4. Left medial paracolic gutter

Pelvic peritoneum

In the male, the peritoneum is related to the walls of the pelvis. Anteriorly it is related to the bladder, posteriorly the rectum and the rectovesical pouch in the lowest region. In the female the uterus and its peritoneal folds divide the pouch into anterior uterovesical pouch and posterior recto uterine pouch (Pouch of Douglas).

Histology of peritoneum

There is a single layer of mesothelial cells (Two types - cuboidal cells and flattened cells). It rests on a basement membrane of loose collagen fibres. The basement membrane overlies a complex highly vascularised connective tissue layers.

D. PHYSIOLOGY

Peritoneal cavity is the largest cavity in the body the surface area of its lining membrane in 1.8 m^2, equal to the surface area of skin. It has been estimated that 1mm increase in thickness of peritoneum by fluid accumulation can result in sequestration of 18L of fluid.

Normally <50ml of sterile, pale yellow coloured fluid is present in the peritoneal cavity. It resembles lymph fluid. It is secreted by visceral peritoneum circulated through the peritoneal cavity, finally the fluid is mostly absorbed into the lymphatic circulation via peritoneal surfaces and also through diaphragmatic lymphatics. Negative intrathoracic pressure during inspiration facilitates this fluid movement into thoracic lymph channel.

Bacterial clearance from peritoneal cavity depends on

1. Sub diaphragmatic lymphatic channels
2. Phagocystosis by peritoneal macrophages.

These two local mechanisms represent the ‘first line’ of clearance after bacterial contamination.

Peritoneum overlaying the muscular portion of diaphragm possesses stomata or intercellular gaps. Both fluid and substances that are not amenable to absorption through

Peritoneal membrane are channeled via the stomata to specialize diaphragmatic lymphatics called lacunae. During inspiration, contraction of the diaphragm empties the lacunae into efferent lymphatic channels and finally via the thoracic duct into systemic circulation.

E. PATHOPHYSIOLOGY

Peritonitis presents with a wide range of pathological change depending on a few factors They are

(i) Source of infection

(ii) Severity of infection

(iii) Age, and general condition and resistance of the host.

(iv) The promptness and efficacy of the method of medical or surgical treatment adopted. Acute diffuse peritonitis is usually infective right from the start where as in peritonitis due to chemical irritation, it may remain non infective for a period of many hours.

When pathogenic bacteria are free to multiply in the peritoneal cavity, the changes that occur are characteristics. The peritoneum becomes hyperaemic and oedematous and fluid is poured out. Initially the fluid in serous and clear, then turbid and finally frankly purulent. This exudate contains fibrin, which helps to localise an infected area by causing coils of intestine and omentum to become stuck together thereby walling off the contaminated parts from the rest of the peritoneal cavity. Free gas in large quantities often accumulates in the abdominal cavity in cases of perforation of stomach or intestine. Small quantities of gas may sometime be produced by gas forming organisms in certain localised intra abdominal abscesses.

Unless localisation has occurred the exudate of peritonitis becomes distributed all over the peritoneal cavity and thus tends to disseminate the infection.

FACTORS INFLUENCING DIFFUSION OF PERITONITIS[21]

(a) Factors favouring localisation of peritonitis.

1. Anatomically the peritoneal cavity proper is subdivided into supracolic, and infracolic compartments by the transverse colon, and its mesocolon. This decreases the spread of infection from one to another.
2. Formation of fibrinous adhesions between the affected organ and parietes
3. Outpouring of serous fluid rich in leucocytes and antibodies.
4. Peristalsis retarded in affected coils and this helps in preventing distribution of infection to other coils.
5. Greater omentum, regarded as the policeman of abdomen envelops the inflammed structures, so as to contain the spread of inflammatory changes.
6. Drains are frequently used post operatively to assist localisation and exit of intra abdominal collections which sometimes helps in containing the spread of inflammation.

(b) Factors predisposing to diffusion of peritonitis

1. Most important factor in precipitating generalized peritonitis is the speed of occurrence.. If a hollow viscus perforates suddenly before protective mechanisms have been mobilised, there in a gush of intestinal contents into the peritoneal cavity which spread over a large area almost instantaneously.
2. Ingestion of food and water helps progressing peritonitis by stimulating peristaltic action.
3. Inadvertent administration of purgatives and enema.
4. Virulence of the organism will make localisation of the infection impossible.
5. Greater omentum is small and less well developed in children, unlike adults.
6. During surgery Injudicious and rough handling of tissue and collections that are localized in specific compartments help the spread of infection.
7. If patient has underlying debilitating diseases immuno compromised status, then the infection can be overwhelming and turn to generalised peritonitis.

Peritoneal healing [22]

Replacement of injured mesothelium occurs over the entire wound surface simultaneously. The rate of the healing is independent of the size of the peritoneal wound. Within 3 days the wound is covered with connective tissue cells and by the day 5, covered by normal mesothelium. The source of these mesothelial cells is unclear, may be arising from sub-endothelial stem cells. Following resolution of inflammation, fibrinous adhesions are degraded and removed. But with severe peritoneal injury or persistent infection, flimsy fibrinous adhesions are transformed to fibrous adhesions by the in growth of fibroblasts, capillaries and deposition of collagen.

Response of Bowel

The bowel response to peritoneal irritation initially is transient hypermotility. After a short interval the motility decreases which progresses to complete adynamic ileus. Bowel distends due to accumulation of air and fluid within its lumen.

Responses of the body fluid compartments.

Peritonitis causes outpouring of plasma like fluid into the peritoneal space as exudate. The atonic bowel also accumulate fluid. This translocation of water, electrolytes and protein into a sequestrated third space functionally removes the volume temporarily from body economy. The rate of functional ECF loss is proportional to the surface area of peritoneum involved in the inflammatory process. With extensive peritonitis, fluid translocation of 4-6L or more in 24 hours in not uncommon.

B. Secondary responses in peritonitis

(i) Endocrine response :

Peritonitis acts as a stimulus to many endocrine organs. There is almost an immediate outpouring of adrenaline and noradrenaline producing vasoconstriction tachycardia and sweating. The adrenal cortex is stimulated to secrete increased amounts of cortical hormones.

Aldosterone and ADH secretion are also increased as a response to hypovolemia in peritonitis, resulting in increased renal conservation of sodium and water. Infact water retention exceeds that of sodium, thus causing hyponatremia.

(ii) Cardiovascular response

Decreased ECF volume results in decreased venous return and decreased cardiac output. The heart rate increases in an attempt to maintain cardiac output. The compensatory mechanism is usually incomplete leading to progressive acidosis which retards the contractility of heart. Thus cardiac output is further reduced resulting in inadequate tissue perfusion and aerobic metabolism at the cellular level.

(iii) Respiratory response

Initially there is an increase in respiratory rate due to hypoxia, and ventilation due to acidosis. Basal atelectasis of the lung is facilitated by abdominal distention and restriction of diaphragmatic mobility which decreases ventilatory volume. Ventilation perfusion mismatching results from both the atelectasis and intrapulmonary shunting due to beta adrenergic stimulation. Pulmonary permeability also increases. So accumulation of fluid in the pulmonary interstitium and alveoli, leading to pulmonary edema, alveolar collapse, eventually adult respiratory distress syndrome (ARDS).

(iv) Renal Response

Decreased cardiac output, increased ADH aldosterone secretion and hypovolemia act synergistically on the kidney. Renal blood flow is diminished resulting in decreased glomerular filtration rate and decreased urine output. Reabsorption of sodium and water are increased. Potassium is excreted excessively. The renal capacity to handle excess solute is impaired. All these favour the development of metabolic acidosis.

(v) Metabolic response

A rapid increase in metabolic rate and oxygen demand of tissues occur simultaneously in peritonitis. The capacity of heart and lungs to deliver oxygen is diminished. Thus poor tissue perfusion leads to a shift from aerobic to anaerobic metabolism at the cellular level. So accumulation of metabolic end product especially lactic acid, causes metabolic acidosis.

The body attempts to compensate with increased respiratory effort to excrete CO_2, but increased respiratory effort in turn places an additional demand in the already inadequate circulation to perfuse muscles of respiration.

Protein metabolism is also altered in peritonitis the protein catabolism begins early. Muscle protein preferentially catabolised leading to weight loss of 25 to 30% lean body mass, if peritonitis persists, plasma protein synthesis especially that of albumin in increased, but circulatory albumin is decreased due to accumulation of albumin in the peritoneal cavity.

C. Specific Responses due to Bacteria

A group of specific responses due to the presence of bacteria are found superimposed on the general response to peritonitis in suppurative peritonitis.

The magnitude of these specific responses is determined by

1. The extent and duration of contamination
2. Presence or absence of adjuvants
3. Virulence of contaminating bacteria
4. Appropriateness of initial therapy

D. Effects of sepsis[23]

The presence of bacteria in suppurative peritonitis leads to a number of both local and systemic responses which are directly related to the effects of the microorganisms or their products. These responses are in addition to and superimposed on the general pathophysiological responses which occur in all cases of peritonitis.

•Toxic effects are due to endotoxins and preformed toxins. Initially it produces a clinical picture of "Warm shock", but when the effects of hypovolemia supervenes, the clinical picture is of "Cold shock".

•Hypovolemic effects

•Respiratory response - pulmonary oedema, loss of pulmonary surfactants, pulmonary collapse and pulmonary consolidation. Finally rapid deterioration of pulmonary function due to the development of "ARDS", "Shock lung", "Septic Lung", "White Lung".

•Renal response - Cortico medullary disconnection, proliferate glomerulo nephritis and acute renal failure.

•Effects on leukocyte function : In severe sepsis both neutrophil and lymphocyte functions are impaired. These effect are reflected in abnormal neutrophil chemotactic responses, decreased ability to lyse the phagosomes and decreased ability of lymphocyte to form rosettes.

E. End organ failure

These multiple deleterious organ effects reinforce one another in a progressively worsening cycle of events. Administration of fluid and electrolytes, respiratory support, reduction of body temperature and maintenance of nutrition all may contribute to survival, but if delayed multiple organ failure ensures. Respiratory, cardiovascular and renal failure develops which progresses to death. So prompt and effective therapy is the most important determinant of survival.

F. BACTERIOLOGY

The causative organisms in primary peritonitis, notably Pneumococcus or Betahaemolytic streptococcus or Gonococcus while most examples of secondary bacterial peritonitis represent the mixed flora of intestinal tract or its adnexae. There is good synergism between the anaerobic and aerobic organisms in this situation.

E.coli and Enterococcus were the predominant organism during the peritonitis phase, while B. fragilis predominated during abscess phase.[24]

Virulence of bacteria is influenced by a number of factors

Capsular poly saccharide components

The size of the bacterial inoculum

The ability to adhere to the mesothelial surface

Bacterial synergism

Adjuvant factors - enhances the virulence of micro organisms by interference with host defense mechanism. Important adjuvant factors are- decreased hemoglobin, decreased fibrin, decreased platelet, increased necrotic tissue, contaminated gastric juice, pancreatic juice, urine, meconium, bile, barium sulfate, talc, drain, suture material, local haemostatic agents.

Routes of bacterial invasion

A. Direct infection

1. Through a perforation of some part of the alimentary tract
2. Through a penetrating wound of the abdominal wall.
3. Post operative

B. Local extension

1. From an inflamed organ eg. - Appendicitis
2. Migration through the gut wall. Eg. Strangulated hernia
3. From the fallopian tubes

C. Blood stream - as a part of general septicemia.

G. CLASSIFICATION

Classification of peritonitis according to etiology[22]

I. PRIMARY PERITONITIS

A. Spontaneous peritonitis in children

B. Spontaneous peritonitis in adult

C. Peritonitis in patient with COPD

D. Tuberculous and other granulomatous peritonitis

II. SECONDARY PERITONITIS

A. Acute perforation peritonitis (Acute suppurative peritonitis)

1. Gastrointestinal tract perforation
2. Bowel wall necrosis (intestinal ischemia)
3. Pelvic peritonitis

B. Post operative peritonitis

1. Anastamotic leak
2. Leak of simple suture
3. Blind loop leak
4. Other iatrogenic leak

C. Post traumatic peritonitis

1. Peritonitis after blunt abdominal trauma
2. Peritonitis after penetrating abdominal trauma

III TERTIARY PERITONITIS

A. Peritonitis without evidence for pathogens

B. Peritonitis with fungi

C. Peritonitis with low grade pathogenic bacteria

Pathological classification

Suppurative peritonitis

Serofibrinous peritonitis

Fibrinous - purulent peritonitis

Faecal Peritonitis

Bilious peritonitis

Hemorrhagic peritonitis

Chemical Peritonitis

Talcum Peritonitis

Classification according to the spread of infection

1. Diffuse peritonitis
2. Localised peritonitis
    - Intrabadominal abscess
    - Interloop abscess
    - Douglas abscess
    - Suphrenic abscess
    - Retrocolic abscess
    - Pancreatic abscess
    - Other abscesses

H. CLINICAL FEATURES AND DIAGNOSIS

Perforative peritonitis is the most common type of peritonitis. The onset of peritonitis may be sudden or insidious. In perforative peritonitis, the onset is sudden and present with classic signs and symptoms of generalised peritonitis.

On non - perforative lesions and in certain post operative cases, the onset is more gradual or insidious. The clinical manifestations of the lesion responsible for the condition gradually merge into those of the first stage of peritonitis.

Depends upon the severity of the lesions peritonitis may be localised or diffused.

Features of localised peritonitis[25]

Patient will have abdominal pain & vomiting. Systemic signs like fever and tachycardia will be present. Important signs like guarding and rigidity of the abdominal wall over the inflamed area will be obvious. Each area will have peculiar presentation, for example shoulder tip pain in subphrenic abscess, urinary symptoms and mucus diarrhoea in pelvic peritonitis. In pelvic peritonitis, abdominal signs are less but tenderness in per vaginal and per rectal examination is more pronounced.

Localised peritonitis when diagnosed early and treated appropriately usually resolves. In about 20% of cases abscess follows. Infrequently localised peritonitis becomes diffuse. Conversely in favorable circumstances, diffuse peritonitis can become localised, most frequently in the pelvis or at multiple sites within the abdominal cavity. A large collection of bile localised to the subphrenic space can remains dangerously. ‘Silent’ until a late stage -

Waltman - Walters syndrome.

Diffuse (generalised) peritonitis

The clinical course can be divided into 3 phases which overlap with each other.

1. Initial phase

(a) Symptoms

Of all symptoms, pain in the most important and constant symptom. It may be either sudden or gradual in onset. It is often severe and continuous in nature made worse by moving or breathing. It is first experienced at the site of original lesion and spreads outwards from this point. There are also atypical presentation. For example, in post operative peritonitis, pain may so mild as to amount to nothing more than discomfort.

Vomiting may be slight at the start but as the peritonitis advances it becomes persistent. In the early stage only the stomach contents are voided. Initially it is reflex in origin. Vomiting may be absent or infrequent if fluid by mouth are withheld.

Bowels are usually constipated although in some cases of pelvic peritonitis there may be diarrhea.

(b) Signs

Temperature changes are variable, but can be subnormal. It may be normal in cases when the onset is sudden. Eg. - perforated duodenal ulcer but it tends to rise gradually as true peritonitis supervenes.

A rising pulse rate and a falling temperature are of the gravest significance, while a gradually rising temperature and a slowly falling pulse rate suggest that localisation of the infection is taking place.

(c) Abdominal examination

Inspection

Abdominal respiratory movement becomes markedly diminished or absent. Decubitus of the patient is typical. He lies very still loathing to move with the legs drawn up in an effort to ease the tension on the abdominal wall

Palpation

Tenderness and rigidity on palpation are typical. The most important sign of peritonitis is guarding and rigidity of the abdominal wall over the area of the abdomen which is involved with a positive ‘release’ sign.

Rebound tenderness i.e. pain caused by sudden release of pressure of the examining hand and tenderness in the affected region elicited by pressure on an uninvolved portion of peritoneum are two other signs.

Auscultation

In peritonitis, Peristaltic sound are diminished from the onset, they may be absent over the causative area.

Percussion

Obliteration of liver dullness is an important diagnostic sign which implies gas under the diaphragm which has escaped into the peritoneal cavity following a leak or perforation.

2. Intermediate phase

Peritonitis may resolve, so that pulse slows, the pain and tenderness diminish, leaving a silent, soft abdomen, which misleads the observer. So this phase is sometimes called as 'stage of delusion'. Sometimes with this phase the peritonitis may become localised with the formation of abscess.

3. Terminal phase

If resolution or localisation have not occurred and peritonitis has progressed for some days this stage is reached. Pulse become rapid, thready and irregular, extremities become cold and clammy with sunken eyes, dry tongue and drawn and anxious face. This is called ‘Hippocratic facies’. There is underlying toxaemia and ileus.

Circulating failure ensures, abdomen becomes increasingly distended, bowel sounds absent and the patient finally lapses into unconsciousness. It is the stage of despair and lost hope. With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice.

I. DIFFERENTIAL DIAGNOSIS

All conditions producing acute abdomen are entertained as differential diagnosis. The following are some of the important conditions which form the differential diagnosis of peritonitis.

1. Thoracic condition
    A) Pleurisy and pneumonia
    B) Pericarditis
    C) Ischemic heart disease
2. Conditions of spinal cord
    A) Tabes dorsalis
    B) Spinal tumour
    C) Herpes zoster
    D) Caries spines
    E) Psoas abscess
3. Diabetes mellitus with ketoacidosis
4. Porphyria
5. Malaria
6. Sickle cell anaemia
7. Haemophilia
8. Renal Disease - acute pyelonephritis
9. Gynaecological condition
    A) Ectopic gestation
    B) Twisted ovarian cyst
    C) Acute salpingitis
10. Intra peritoneal haemorrhage

Surgical condition that should be differentiated from perforative peritonitis are

1) Acute appendicitis

2) Acute pancreatitis
3) Intestinal obstruction
4) Mesenteric thrombosis or embolism
5) Acute cholecystitis
6) Acute diverticulitis
7) Acute salphingitis
8) Dissecting or ruptured aneurysm of abdominal aorta
9) Ruptured ectopic gestation
10) Acute pyelonephritis
11) Primary Peritonitis

J. INVESTIGATIONS

AIMS

1. To confirm the diagnosis
2. To determine the cause
3. To study the microbiological aspects
4. To assess the biochemical changes of body fluids.

URINE

1. Hourly urine output measurement
2. Urine routine examinations like urine albumin sugar and deposit

BLOOD

1. **Hemoglobin percentage and Hematocrit.**
2. **Total Count and DC -** usually shows polymorpho nuclear type of leukocytosis
3. **Erythrocyte sedimentation rate**

ESR will be high in cases of acute diffuse peritonitis. Very high values are seen if the underlying cause is tuberculosis or malignancy.

4. Blood chemistry

Blood urea, Serum creatinine, serum electrolytes, will help to assess and correct disturbance in individual cases and used in modified APACHE II were recorded.

5. Blood culture and others

Blood culture is relevant in cases of septicaemia and typhoid fever.

Widal reaction, liver function, test etc. are done in relevant cases.

RADIOLOGICAL EVALUATION[26]

1. Plain x-ray chest - to rule out chest pathology

2. Plain x-ray abdomen - erect posture - may show free gas under diaphragm in hollow viscus perforation. This can also be demonstrated in lateral decubitus view. Free air can be demonstrated in 75-80% of perforations only, so its absence does not absolutely rule out perforation.

3. Contrast radiography - In doubtful cases with no free air under diaphragm, administration of gastrograffin (water soluble contrast medium) will reveal the site and extend of perforation.

4. Ultrasound examination

Shows free fluid in the abdominal cavity or localised fluid in the peritoneal spaces.

5. CT

Most sensitive test for perforation but it is rarely required. It shows both fre air and localised collections

DIAGNOSTIC PERITONEAL ASPIRATION

A positive tap is useful and give valuable information, where as a negative tap is not significant.[27]

The aspirated fluid is studied for its physical and chemical characteristics. It can be stained for bacteria, examined under microscope and can be cultured.

K. MANAGEMENT

The primary objectives in the management of secondary peritonitis are

1. Resuscitation and general care of the patient
2. Antibiotic therapy
3. Surgical management
4. Post operative management

1. Resuscitation and general care of the patient

a) Intravenous fluids

In peritonitis, large amounts of fluid and electrolytes may be lost. Treating circulatory shock, if present should be given prime importance. The deficit and ongoing loss of fluid can be replaced by administering intravenous fluids, often in large volumes. This includes crystallized solution, for lost water and electrolyte, whole blood or packed cell to correct anaemia.

The effectiveness of therapy is judged by the normalisation of pulse, blood pressure, mental status and urine output. Central venous catheter should be placed in patient with septic shock, old age or cardiac, pulmonary and renal insufficiencies.

b) Nasogastric aspiration

Ryles tube is passed into the stomach to decompress stomach, and intestinal. This will prevents pulmonary aspiration, abdominal distension by reducing the accumulation of air in the paralysed loop. Intermittent aspiration is maintained till the ileus is resolved.

c) Urinary catheterisation

To monitor urine output. The output should be maintained at 30-60 ml/hour.

d) Analgesia

Pain must be relieved before and after the surgery. Morphine may be given and

continued for 48 hours.

e) Oxygen and ventillatory support

A nasal catheter delivering oxygen can be used to correct mild hypoxia. If their is an impairment of ventilatory volume arterial blood gases are to be measured. If the arterial partial pressure of oxygen is below 70 mm of Hg, ventillatory support with inspired gas concentration of 40% oxygen is administered. If PO_2 in below 60 mm of Hg or in those who show severe respiratory embarrassment, addition of P.E.E.P to ventillatory support is indicated.

f) Monitoring vital signs

Pulse rate and volume, blood pressure, temperature, respiratory rate, CVP, urine output, arterial gas analysis etc. are continuously monitored for effective management of the patient.

g) Others

(i) Vasoactive drugs - when volume - replacement fails to restore the circulation, administration of Dopamine is helpful. It has a potent ionotropic and chronotpic effect on the heart

(ii) Antipyretics - Fever is treated by antipyretic agent like Paracetomol, tepid sponging. In case of hypothermia slow warming with blanket is done.

(iii) H2 Blockers - to reduce acid secretion.

2. Antibiotic therapy

Administration of antibiotics prevent multiplication of bacteria and the release of endotoxins. As the infection is usually a mixed one, presumptive therapy which covers aerobic gram negative rods and anaerobic organism is started. The agents commonly used against aerobic organisms are aminoglycoside, II & III generation cephalosporine, monobactum, ampicillin with sulbactum / clauvulanic acid etc.,

For anaerobic organism metronidazole is used. To prevent the recurrence of sepsis, therapy should be continued for adequate duration, i.e. till the temperate and cell count become normal.

3. Surgical Management

In perforative peritonitis, surgical control of the infecting organism is the main stay of treatment. Here, only basic general surgical concepts are discussed, specific operations for each organ is dealt with in the later part.

Aims of operative management are

1. Control of source of contamination
2. Reduction of bacterial inoculum

i) Control of source of contamination

Once the patient is fit for anaesthesia and surgery, exploration of the abdomen is carried out through a carefully planned incision. If the diagnosis is doubtful, a midline vertical incision is used which gives access to majority of the peritoneal cavity. The contaminating source is identified and dealt with simple closure of perforation or resection of the perforated viscus or exclusion of the affected organ.

ii) Reduction of bacterial Inoculum.

The following procedures are generally advocated

a) After the cause has been dealt with whole peritoneal cavity is explored, the collected fluid is sucked out, debridement and removal of fibrin, blood clot and necrotic material is done. Copious irrigation of peritoneal cavity with 2-3 litres of normal saline added either with antibiotic or antiseptic agents is carried out. This decreases the mortality and post operative infection.

4. Post operative management

Aims

1. Relieve post operative pain
2. Maintain fluid and electrolyte balance
3. Nutrition of the patient
4. Reduces abdominal distension.
5. To ensure early recovery, especially paralytic ileus.
6. To prevent, detect and treat post-operative complications

Measures

1. Relief of pain by narcotic analgesics like morphine, pethedine or pentazocine
2. Fluid intake output chart is maintained. The usual requirement of fluid along with insensible and ongoing loss of fluid is supplemented. Depending on the amount of ryles tube aspirate, urine output and CVP adequate hydration is assured.

   Serum levels of sodium, potassium, and calcium etc., are estimated and supplemented accordingly.
3. The Caloric requirement is met with by putting a central venous catheter into the SVC and giving 10% glucose.
4. Continuous nasogastric aspiration will give decompression and rest to the intestines. Signs of recovery are seen by 3rd and 4th post operative day.
5. Broad spectrum antibiotics are continued and suitably changed accordingly to culture and sensitivity report

6. Vital signs are continuously monitored during the immediate post operative period.

Drain is removed depending on the assessment of drainage. Ryles tube is removed when the patient has good appetite and has passed flatus faeces and also when aspirate decreases with appearance of peristatic sound. Oral fluids can be started gradually.

INDIVIDUAL PERFORATION AND ITS MANAGEMENT

Peptic Ulcer Perforation

Perforation secondary to peptic ulcer may be either gastric or duodenal ulcer perforation which again can be acute or chronic.

BONNEEVIE in 1985 states that 7% of all known cases of duodenal ulcer perforate and in 2% of patients this is the first manifestation. 80% of the patients have the history of peptic ulceration and the other 20% have silent chronic ulceration. Among the duodenal ulcer anterior perforation constitutes 92%, posterior perforation 2% in the posterior part of duodenum and pyloro duodenal junction 6%. Usually ulcer in the anterior wall perforates and in the posterior wall penetrate with severe bleeding. In 5-10% of the cases may be of the kissing ulcer occurring both on the anterior and posterior wall.

In gastric ulcer those present on the anterior wall of the stomach and the lesser curvature and ulcers over the pyloric antrum perforate frequently than the posterior wall.

Multiple perforation and ulcers in the stomach duodenum and jejunum in a person should alert the physician about Zollinger Ellison Syndrome.

Surgical treatment

The peptic ulcer perforation

It should be operated on as early as possible, delay in treatment especially more than 24 hrs increases the mortality, morbidity and length of hospital stay.

Usual approach is through a supra umbilical midline incision. Bile stained fluid with fibrinous exudate suggests the diagnosis. If the anterior perforation is not found, the lesser sac should be entered and searched for posterior perforation.

- **Perforated duodenal ulcer**

Should be closed with full thickness, single layer interrupted, absorb able sutures, reinforced with live omental onlay (CELLAN - JONES - 1929). If the perforation is large with oedematous edge, it is closed with island omental patch (ROSCCOE : GRAHAM : 1933). All simple closure procedure should be followed post operatively with anti secretary drugs for 6 months after healing of the ulcer.

Role of definitive acid reductive surgery

Proximal gastric vagotomy is routinely advised (Boey and colleagues - 1982) in all cases except in the following conditions.[28]

1. If the patient in haemodynamically unstable
2. Perforation more than 24 hours
3. Gross peritoneal contamination with food and purulent maternal.

These procedures reduce the recurrence of ulcer and perforation and subsequent need for reoperation. Of late with the advent of potent proton pump inhibitors, the acid reduction surgery can be foregone.

- **Perforated gastric ulcer** - the options are

1. Simple closure and four quadrant biopsy of the ulcer
2. Excision and primary closure
3. Gastric resection

The choice of operation is also influenced by the age of the patient, location of the ulcer, general condition of the patient, degrees of peritoneal contamination and the presence of malignancy on frozen section biopsy

For example

- For ulcers in distal stomach, Antrectomy with GJ serves both removal of the ulcer and provides definitive therapy.
- For benign ulcers in unstable elderly patients excision and closure with omental patch.
- For ulcers in the lesser curvature excision and closure is advocated.

Patient with the following entities require a definitive ulcer operation

1. Perforated gastric ulcer
2. Combined gastric and duodenal ulcer, one of which has perforated
3. Perforation with pre existing chronic ulcer symptoms
4. Coexistent obstruction and perforation
5. Coexistent hemorrhage and perforation
6. Previous operation for perforated duodenal ulcer

Non operative management of peptic ulcer perforation

According to Donovan non operative management should be reserved for[29,30]

1. Patient who have perforation of longer than 24 hrs duration
2. For patient whose systemic disease or current state preclude operative treatment
3. Sealed perforation
3. Water soluble contrast study shows no free leak into the peritoneal cavity.

Management includes

1. Nasogastric suction
2. Intravenous fluid replacement
3. IV H_2 blockers and antacid therapy
4. Intensive antibiotic treatment
5. Close clinical observation

These patient may develop intra peritoneal abscess which can be managed with percutaneous catheter drainage.

Laparoscopic surgery in perforated ulcer[31]

Perforation is closed with intracarporeal suturing in a manner identical to the open surgery reinforcement with omentum can be done. Following the procedure abdomen thoroughly irrigated and aspirated. Depending upon the surgeons experience definitive procedures like proximal gastric vagotomy or taylor procedure (Anterior seromyotomy with posterior truncal vagotomy) canbe performed.

Traumatic Perforation and other causes

The incidence of abdominal trauma continues to increase with modernisation of this world. Traumatic perforation could be either due to blunt injury or penetrating injury. The frequency of intestines being involved in abdominal trauma is about 16%.

Stomach

Penetrating injuries are more common than the blunt injuries because of the relative lack of fixation of the stomach. Apart from the classical picture of perforation patient may have blood aspirated through the Ryles tube. On exploration both the anterior and posterior wall should be searched thoroughly. The rent is closed in two layers.

Duodenum[32]

Injuries to the duodenum and intestine comprises about one quarter of the abdominal trauma. Retroperitoneal part of duodenum is frequently involved and cause chemical peritonitis because of high alkaline pH of this duodenal content. Testicular pain should raise the suspicion of retroperitoneal rupture of the duodenum. Large accumulation of air above the right kidney in x-ray and CT is diagnostic.

Surgical treatment depends upon the size of perforation. If it is small, simple closure is enough. In large perforation simple closure will cause stricture. So one of the following procedure can be adopted.

A) Complete division of duodenum and end to end anastomosis.
B) Division of duodenum, closure of both ends and a gastro enterostomy.

Small intestine

In traumatic perforation commonest cause of disruption is blunt injury abdomen. Rupture occurs at the point where the fixed part joins the mobile one. Other causes are obstruction, typhoid, tuberculosis, ischemia, malignancy, diverticulosis, crohns disease etc.

Large intestine[33]

Traumatic rupture is less frequent in the large intestine. In ulcerative lesion of the rectum, an insufflation of air is sufficient to perforate the lumen. Other causes for perforation are inflammatory bowel diseases, diverticulosis, malignancies, etc. In colonic perforation wide spread contamination with faecal peritonitis commonly occur. The basic principle in the management of coloric perforation is earliest possible elimination of the perforated segment. The following procedures are recommended.

a) Exteriorisation of the affected segment
b) Excision and endcolostomy with mucus fistulae
c) Hartmann's procedure
d) Resection and end to end anastomosis.

Enteric Peforation[34]

It occurs during the third week. Sometimes this may be the first presenting feature. Here the ulcer is parallel to the long axis of the gut and usually present in the distal part of the ileum.

Intestinal Obstruction

In the intestinal obstruction perforation occurs just proximal to the obstruction. Depends upon the size, simple closure or resection and anastomosis is done.

Gallbladder perforation

The Gallbladder perforation is usually due to gangrenous acute cholecystitis which is treated by cholecystectomy which is uncommon in our geographical area.

Appendicular perforation[35]

The appendix may perforate at any spot, but most frequently along its anti mesenteric border. Commonest cause of its perforation is faecolith obstructing the lumen. Following perforation, either localised abscess is formed in the right iliac fossa or in the pelvis or end up in diffuse peritonitis. Poor localization occurs in the extremes of ages. The localized collection of pus may be aspirated with ulstrasound or CT guidance. Extraperitoneal drainage can be attempted in some cases. In acute perforation with diffuse peritonitis appendisectomy with through peritoneal lavage and drainage of the area is advised.

L. COMPLICATIONS[36]

All the complications of a severe bacterial infection are possible but the specific complications of peritonitis are as follows.

1. Residual abscess[36] - in majority of cases the abscess resolves with antibiotic therapy. If the abscess fails to resolve, it can be managed by percutaneous or opens drainage.
2. Paralytic ileus
3. Acute intestinal obstruction due to peritoneal adhesions
4. Wound infection - infection is more in intestinal perforation
5. Wound dehiscence and burst abdomen
6. Fistulae due to anastomotic leakage
7. Deep vein thrombosis
8. Pulmonary complications - Bronchitis, Atilectasis, Pneumonia, Pulmonary Embolism
9. Other complications - vomiting, Hiccup, parotitis, urinary retention.

M. PROGNOSIS

With modern treatment perforative peritonitis carries a mortality range from 10% to 40%. Mortality for duodenal ulcer and appendicular perforation is usually normally about 0 to 10%, for intestinal perforation 20 to 40% and for post operative perforation about 30%. The factors influencing mortality are

a) Age of the patient : Greater in the older age group

b) Time interval between the occurrence of perforation and initial treatment. There is approximately five fold increase in the mortality among the patients who received the treatment after 24 hours compared to patients who reached within 6 hours.

c) Site of perforation : Mortality is more in colonic perforation.

d) Extent of disease

e) Electrolyte imbalance

f) Undrained collections

g) Multisystem breakdown - Renal, Cardiac, hepatic and pulmonary insufficiencies

h) Malignancy, diabetes, etc.,

MATERIALS AND METHODS

A prospective survey of patients with acute generalized peritonitis due to gastro intestinal perforation was carried out in general surgical wards of Coimbatore Medical College hospital, Coimbatore during the period starting from January 2005 – December 2005.

The study population consisted of 50 consecutive patients who had who had laparotomy during the study period for acute peritonitis due to gastro intestinal perforation.

The case detection was done on the following criteria.

Inclusion Criteria.

1. Adult patients with features of Acute perforative peritonitis.
2. Patient whose plain x-ray abdomen showed features of hollow viscera perforation peritonitis.
3. Patient with blunt of penetrating injury of the abdomen with signs of hollow viscus perforation.

Exclusion Criteria

1. Patient who presented with features of peritonitis and had no evidence of perforation radiologically and per operatively.
2. Patients with post operative peritonitis.
3. Patient with iatrogenic perforation during laparotomy or endoscopy
4. Patient with esophageal perforation
5. Perforative peritonitis in paediatric age group.

Methods

All patients were evaluated clinically, hematological and bio chemical investigations were carried out. Patients were resuscitated with intravenous fluids and correction of electrolyte imbalance as indicated by the results of the electrolytes and urea.

X-ray – Plain X-Ray abdomen Erect

- Plain X-ray chest PA View done.

The following Acute physiological parameter of APACHE II were assessed and recorded at the admission point preoperatively.

Temperature (degree centigrade)
Mean Arterial blood pressure (mmHg)
Heart rate, Respiratory rate (non ventilated)
Serum Sodium (mMol/1)
S. Potassium (mMol/l)
S. Creatinine (mg / 100 mm)
Hematocrit (%)
White blood count (total / cm^3)
HCO_3 (mmol/l)

No patient had arterial pH or partial pressure of oxygen (Po_2) due to lack of facility.

These were scored in accordance with the Modified APACHE II chart, scoring the abnormality high or low levels.

The scores ranged from 0 to 4 on each side of normal value. Zero represents normal values and increase to 4 indicating the extreme end of high or low abnormal values. These parameters represent the acute physiological scores (APS).

Included in this study as part of APS was the serum urea. This was scored using the parameter similar to that of serum creatinine.

Age points are as follows for adult patients.

44=0, 45-54 = 2, 55-64 = 3, 65-74 = 5, 75=6

Chronic ill health value was added if the patients has history of lever organ system insufficiency or is immuno compromised points are assigned as discussed earlier.

The Sum total of the APS, Age point and chronic health values is the total modified APACHE II Score.

All the parameters were entered in the Modified APACHE II Table as discussed earlier.

Abdominal paracentesis done and specimen sent for culture and sensitivity.

After proper clinical assessment the patients were actively resuscitated with intravenous fluids, nasogastric aspiration, antibiotics, analgesics. A combination of ampicillin, gentamycin & metronidazole were used initially in all cases. Antibiotics were later changed according to the culture and sensitivity report. The bladder was catheterised to monitor the urine output.

After stabilising the general condition, the patients were taken up for surgery. Surgery in the form of laparotomy was done under general or epidural anaesthesia in the majority of cases. The incision used depended on the suspected site of pathology. Most of the cases midline incisions were used, viscera were inspected carefully, the site of lesion located and the appropriate surgical procedure was performed.

Peritoneal toilet and lavage with normal saline were carried out and the peritoneal cavity drained. The abdomen was closed in layers or by mass closure using No. 1 prolene.

Post operatively nasogastric aspiration, antibiotics were continued, nutrition and electrolyte balance were maintained with intravenous fluids. Daily patients were assessed for recovery and complaints if any were recorded.

A separate proforma for each case, containing all the relevant particulars were maintained and all cases were followed up throughout the postoperative period. Specific instruction was given to each patient on discharge, to come for periodical review regularly.

OBSERVATIONS AND RESULTS

I have studied 50 consecutive patients having Acute perforative peritonitis admitted in general surgical wards during the period of January 2005 to December 2005.

Clinical diagnosis was made from history, physical examination and investigations.

Depending on the general conditions of the patient, the line of management was planned.

Exploratory laparatomy was instituted in all cases. Pre – operative resuscitation was done before laparotomy was attempted in all cases and primary causes treated accordingly.

Out of 50 patients 50 underwent laparotomy.

Age and Sex Distribution

Table 1 shows that perforation was common in 31-40 in our study, especially due to duodenal ulcer perforation.

Male to Female ratio was 5 : 1

Table – 1

AGE DISTRIBUTION

Age Group	No. of Patients	Percentage
13-20	7	14
21-30	7	14
31-40	16	32
41-50	9	18
51-60	7	14
> 60	4	8

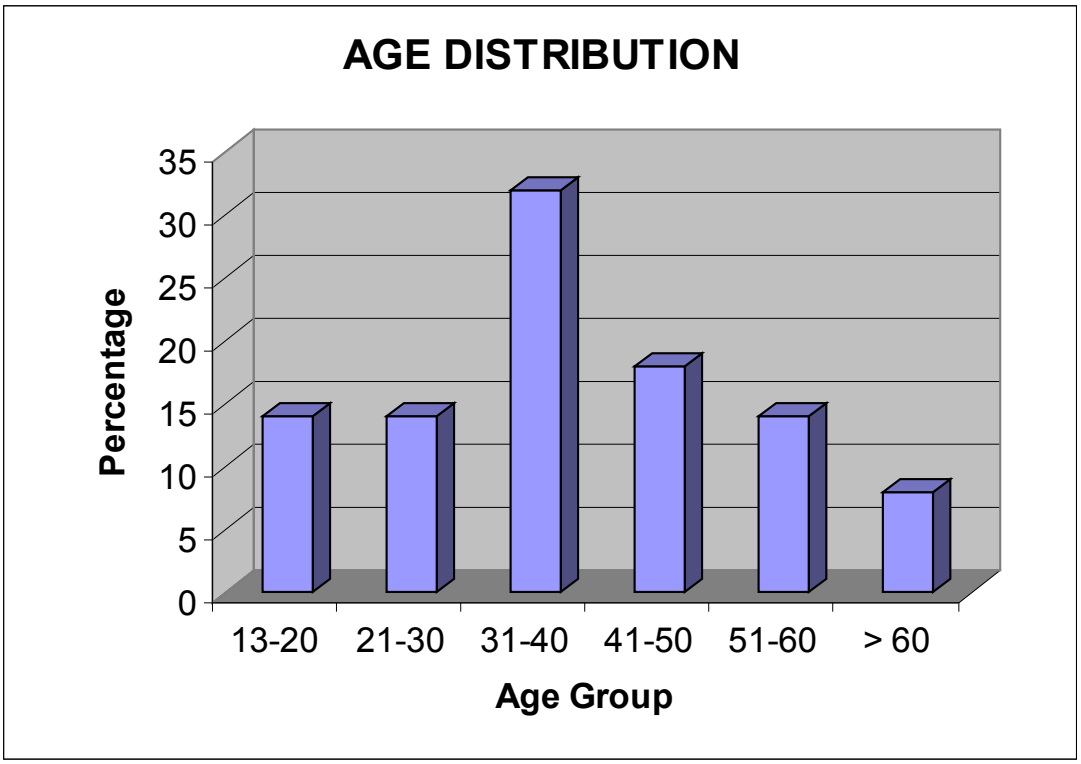


Table – 2

TIME OF PRESENTATION

Time in hrs	No. of Cases	Percentage
0 – 6	8	16
6 – 12	6	12
12 – 24	16	32
24 - 48	16	32
>48	4	8

Only 8 patients got admitted within 6 hours.

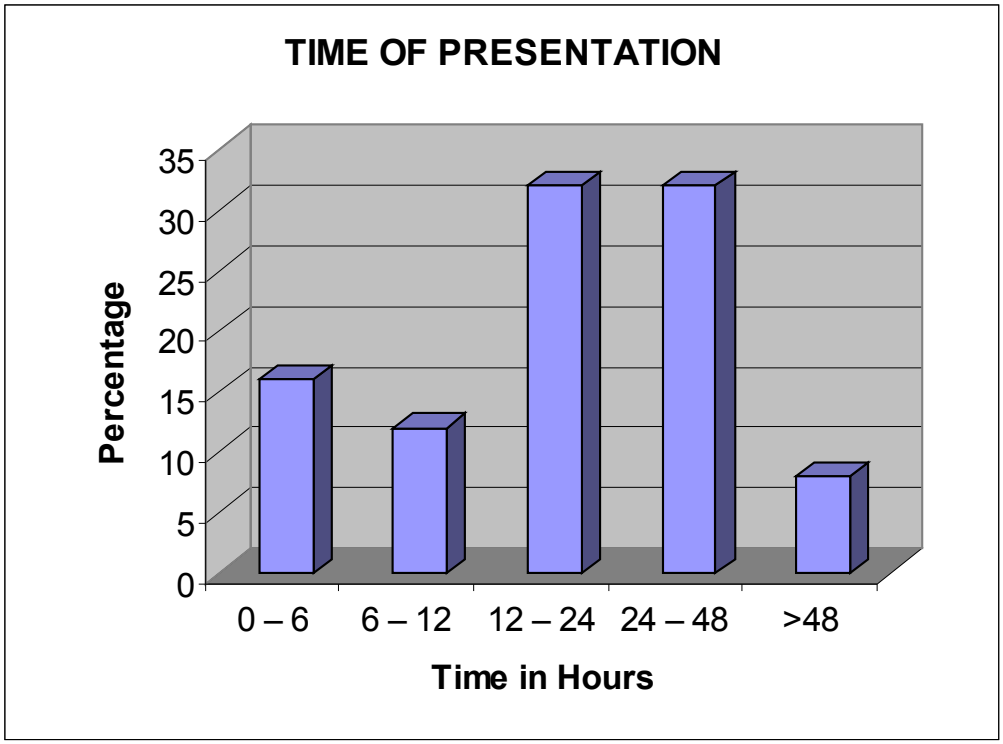
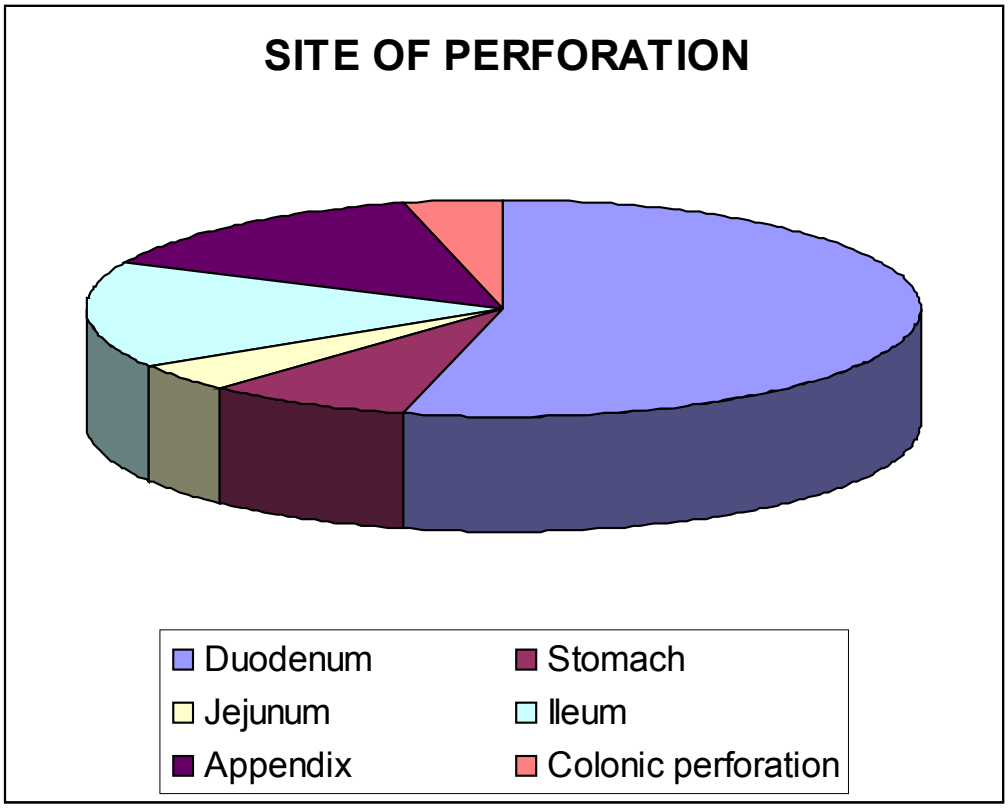


Table - 3

SITE OF PERFORATIONS

Site of Perforation	No. of Cases	Percentage
Duodenum	27	54
Stomach	4	8
Jejunum	2	4
Ileum	8	16
Appendix	7	14
Colonic perforation	2	4



Commonest site of perforation was in the 1st part of duodenum

Duodenal ulcer constitutes the most common cause of gastrointestinal perforation. 75% of duodenal ulcer patients give a history of previous peptic ulcer diseases.

Among the 4 gastric perforations one had malignant perforation who underwent gastrectomy later. Ileal perforations were 8 there were 4 cases due to trauma and other due to enteric fever which was subsequently proved by investigations.

2 patients had jejunal perforation in this study. Both were due to trauma. Appendicular perforations were seen in 7 cases. Appendix was found to be gangrenous in all cases.

In our study there were two cases of colonic perforation which were due to malignancy.

CLINICAL FEATURES

Table - 4

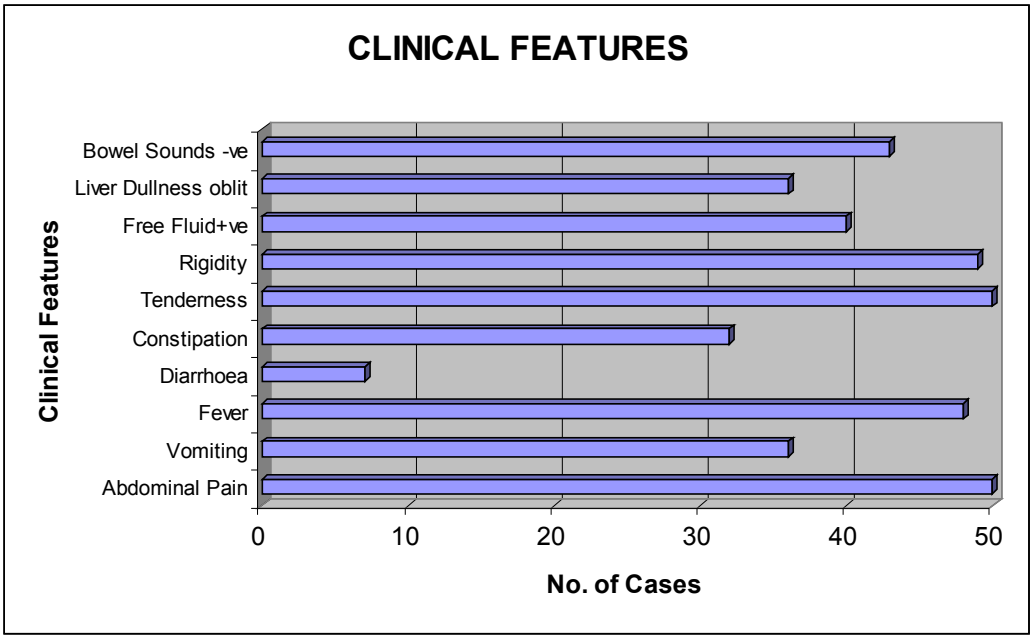
Analysis of symptoms in relation to aetiology

No.	Aetiology	Clinical Features					Total No. of Cases
		Abdominal Pain	Vomiting	Fever	Diarrhoea	Constipation	
1	Duodenal	27	18	26	2	18	27
2	Gastric Ulcer	4	3	3		4	4
3	Jejunal	2	2	2		2	2
4	Ileal	8	5	8	3	4	8
5	Appendicular	7	6	7	2	2	7
6	Colonic	2	2	2		2	2
		50	36	48	7	32	50

Table - 5

Analysis of various signs in relation to aetiology

No.	Aetiology	Clinical Features					Total No. of Cases
		Tenderness	Rigidity	Free Fluid +ve	Liver dullness obliterated	Bowl Sounds -ve	
1	Duodenal	27	27	25	25	24	27
2	Gastric Ulcer	4	4	4	3	4	4
3	Jejunal	2	2	1	1	2	2
4	Ileal	8	8	5	4	6	8
5	Appendicular	7	6	4	2	5	7
6	Colonic	2	2	1	1	2	2
	Total	50	49	40	36	43	50



The table 4 and 5 gives various symptoms and signs in relation to etiology.

Abdominal pain is the commonest.

Vomiting was present in 36 patients – bilious in nature.

Table gives various signs in relation to etiology.

Rigidity was found in 98% of cases.

Liver dullness was obliterated in 72% of cases. Absence of bowel sounds was seen in 86% of patients.

INVESTIGATIONS

Since the diagnosis of peritonitis was many a time clinically obvious and the stage at which they reached the hospital gave very little time for investigations, the spectrum of investigation was limited. But all the routine basic investigations were done. Ultrasound abdomen was done in very few cases with suspicion of localized collection of fluid intra abdominaly. Contrast study was not done in any of our patient.

The most rewarding investigation was plain x-ray abdomen erect view which showed the following findings, gas under the diaphragm, ground glass appearance, distended bowel loops. Gas under diaphragm was present in 74% of cases, especially gastric, duodenal and colonic perforation. It is not a reliable investigation in appendicular perforation.

Diagnostic paracentesis was positive in 84% of cases. The most common organism was Escherichia Coli.

Table - 5

BACTERIAL ISOLATES

Bacteria isolated	No. of Cases
Escherichia coli	29
Klebsiella pneumonia	2
Proteus Vulgaris	1
Psuedomonas	
Staphillococci	
Anaerobic Bacteria	10

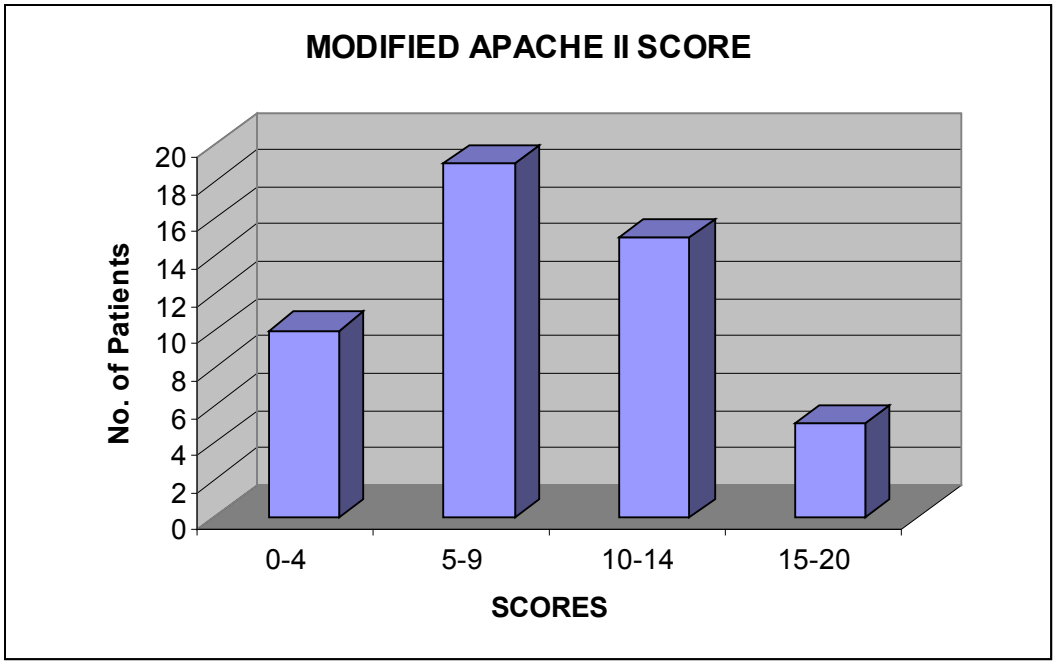
1. Positive culture isolated – obtained in 84% cases

2. Negative culture isolated – obtained in 16% cases.

Table - 6

Modified APACHE II Scores observed in our study.

No.	Etiology	Modified APACHE II Scores				Total Cases
		0-4	5-9	10-14	15-20	
1	Duodenal	9	12	4	2	27
2	Gastric	-	1	3	-	4
3	Jejunal	-	-	1	1	2
4	Ileal	-	1	5	2	8
5	Appendicular	1	5	1	1	7
6	Colonic		-	1	-	2
	Total	10	19	15	6	50



TREATMENT

All patients were takenup for laporatomy after adequate resuscitation with intravenous fluids, nasogastric suction etc.,

Laparotomy was done either through midline or right para median incision. Peritoneum was found to be thickened and there was increased amount of fluid in the peritoneal cavity. The nature of fluid vary according to the aetiology, site of perforation, and time interval between perforation and laparotomy. The cases which reached the hospital early had only minimal collection in the peritoneal cavity. All collections in the peritoneal cavity was sucked out and debridement of necrotic materials was done.

The essential mode of treatment in peptic ulcer perforation was by simple closure, either with live omental patch or with island omental patch. The perforation were closed with 2-0 vicryl in a single layer. Whereas small bowel perforation was closed with two layers inner all coat layer with absorbable suture material and outer seromuscular layer using non absorbable material. Care was taken to avoid tension along the suture line.

Since colonic perforation were due to malignancy, right hemi colectomy was done in one, proximal colostomy and Hartmann's procedure was done in the second case. Definitive surgery for peptic ulcer was done in one case. Here gastro jejunostomy with truncal vagotomy done along with closure of perforation.

After closure of perforation, complete peritoneal lavage was done with 2-3 litres of normal saline. No antibiotic solution was used for lavage at the time of surgery, but in badly contaminated cases metronidazole was instilled just before closure.

The abdomen was closed in layers with a drain in the flank. In very high risk patients peritoneal lavage was done by introducing malecots catheter in both flanks.

The appendicular perforation were dealt with mostly by right para median incision. Appendiectomy was done in all the cases, but the appendicular slump was buried only in two cases because of caecal oedema. In one case appendicectomy was done by retrograde method.

POST OPERATIVE COMPLICATIONS

Respirator infection was found in 10 patients. Wound infection in 16, Intra peritoneal abscess in 3, fecal fistula in 2, wound dehiscence in 2.

Table – 7

POST OPERATIVE COMPLICATIONS

No	Aetiology	Respiratory Infection	Wound Infection	Intra peritoneal abscess	Fecal Fistula	Wound Dehiscence	Total No. of Cases
1	Duodenal	8	8	1		1	18
2	Gastric	1	1				2
3	Jejunal	1				1	2
4	Ileal		2	1	2		5
5	Appendicular		4	1			5
6	Colonic		1				1
		10	16	3	2	2	33

Table – 8

MODIFIED APACHE II SCORE AND POST OPERATIVE COMPLICATIONS

	APACHE Scores				Total No. of Cases
Post operative complications	0-4	5-9	10-14	15-20	
No. of cases with complications	6	14	8	5	33

The mean number of days for hospital stay in patients with post operative complications were higher. The complications were treated according to the nature of the complications.

MORTALITY

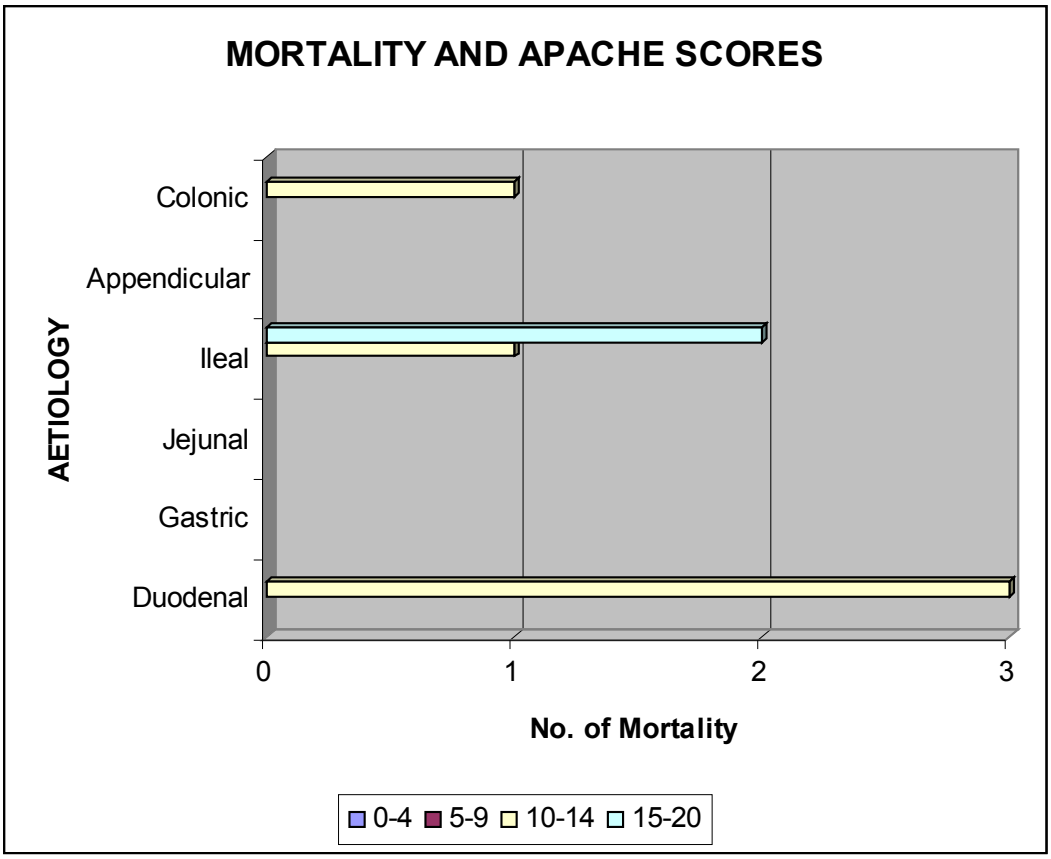
The total mortality was 7 among 50 patients. Three in duodenal ulcer perforations, 3 in small bowel perforations, and one in colonic perforation. The Mortality were very high in the group of 10 - 14 and 15 – 20 range of modified APACHE scores.

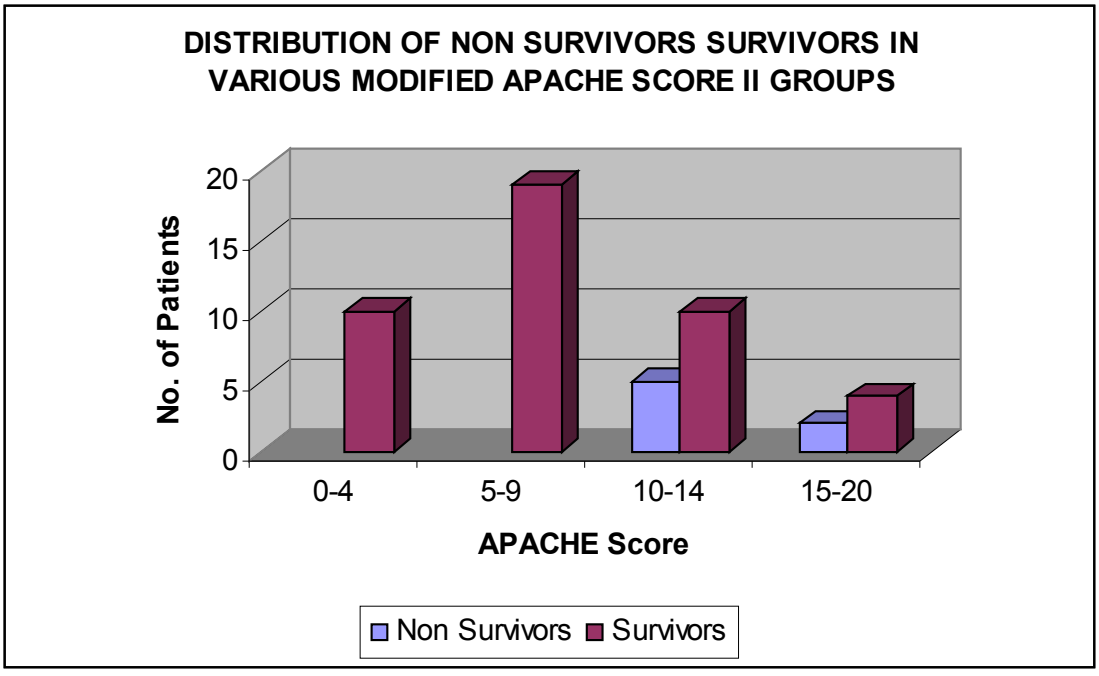
Table - 9

MORTALITY AND APACHE SCORES

No.	Aetiology	Death and APACHE SCORE				Total
		0-4	5-9	10-14	15-20	
1	Duodenal			1	2	3
2	Gastric					0
3	Jejunal					0
4	Ileal			1	2	3
5	Appendicular					0
6	Colonic			1		1
	Total			3	4	7

Causes for mortality were septicemia and electrolyte imbalance.


MORTALITY AND APACHE SCORES
AETIOLOGY
Colonic
Appendicular
Ileal
Jejunal
Gastric
Duodenal
0
1
2
3
No. of Mortality
0-4
5-9
10-14
15-20


DISTRIBUTION OF NON SURVIVORS SURVIVORS IN VARIOUS MODIFIED APACHE SCORE II GROUPS
No. of Patients
20
15
10
5
0
0-4
5-9
10-14
15-20
APACHE Score
Non Survivors
Survivors

ANALYSIS

METHODOLOGY

Mean and standard deviation of the total modified APACHE II Score was compared for each of the complication and mortality for the study.

't' – test was used to compare the statistical significance of the mean values, p value < 0.05 was considered as statistically significant.

Table – 10

STATISTICAL ANALYSIS

Post operative outcome	Mean		Standard Deviation	't' values	'p' values <
Resp. Infection	-VE	8.00	3.64	0.911	0.367
	+VE	6.80	3.64		
Wound Infection	-VE	7.70	3.62	-0.40	0.968
	+VE	7.75	3.784594		
Intra peritoneal abscess	-VE	7.675	3.7	-0.299	0.767
	+VE	8.333	3.214		
Fecal fistula	-VE	7.58	3.66	-1.109	0.274
	+VE	10.50	2.12132		
Wound dehiscence	-VE	7.58	3.53	-1.109	0.274
	+VE	10.500	6.3639		
Death	-VE	7.7209	3.64055	-5.799	0.001
	+VE	16.1429	2.96808		

Modified APACHE II score ranged from 3-20

In morbidity parameter

For Respiratory infection, mean having infection 6.8 + 3 not having resp. infection 8.00 + 3.64, p < 0.367.

Wound infection mean for positives was 7.75 + 3.78 negatives was 7.70 + 3.62, p < 0.968.

In the abdominal abscess mean for positives was 8.33 + 3 negative was 7.6750 + 3.7, p < 0.767

Fecal fistula mean for positives was 10.500 + 2.12 negative 7.58 + 3.66, $p < 0.274$

Wound dehiscence mean for positive was 10.50 + 6.36 negatives was 7.58 + 3.58, $p < 0.274$.

The mean APACHE II Score for survivors was 7-72 $\pm$ 3.6

Non survivor was 16.1429 + 2.9 $p < 0.001$

In this study it was observed that there is a increase in mean apache scores for patients having severe post operative complications like intraperitoneal abscess, fecal fistula and wound dehiscence. This study helps to identify high risk groups where severe morbidity can be expected.

Higher modified APACHE II scores statistically influenced mortality in all the patients irrespective of aetiology with $p < 0.001$ which is statistically significant..

DISCUSSION

Duodenal ulcer perforations were more common, in the age group 31-40, in our study when compared to Devitt Taylor and Debakey's above 60 years and 50-60 years respectively.

Male : Female ratio was 5 : 1 in our study.

Table - 11

Study	M : F
Andrew M Desmond	6 : 1
Rodney Maingot	6 : 1
Our Study	5 : 1

Compared to western studies Crawford and Ellis (1985)[37] it was found that large bowel perforations in our study are lower compared to Western population.

Our study had similar distribution that of previous Indian studies Kachroo et al [38] and Sharma et al., [39] which showed common etiology being Duodenal ulcer, ileal and appendicular perforation in order of frequency.

E.- Coli was the predominant organism in culture in our study.

Morbidity was observed in 66 percent of patients, mortality was 7 in 50 i.e., 14% which is accepted mortality. Maingot et al (10-40%).

Etiology wise duodenal ulcer patients had very low mortality 3 out 27, whereas colonic perforation and enteric perforation had high mortality.

APACHE II Parameters have been shown to have stronger relationship to the outcome then previous grouping such as anatomy, causes, abnormality, age and chronic ill health without consideration for systemic effects of the intra abdominal sepsis.[40] Thus its use in this study.

The APACHE II score is very popular and has been used in both surgical and non –

surgical patients, it has also been validated using many patients over several years in many centers in the developed countries.[1,4,5,6,18,41.] The limitation of the study was lack of facilities for doing ABG.

The Modified APACHE II score for the morbidity for the patients having severe complications like abdominal abscess, fecal fistula, wound dehiscence, were higher but were not statistically significant. This may be due to the cross sectional nature of our study and the sample size. They helped to identify high risk groups where higher complications can be expected.

Table – 12

MORTALITY AND MEAN APACHE II SCORES

Study	Modified APACHE II SCORES Mean	
	Survivors	Non Survivors
Adesunkanmi et al[1,18]	7.6 + 4	9.4 + 2
Our Study	7.72 + 3.6	16.14 + 2.96

In mortality, higher APACHE II Scores were noted. There was no death in scores ranging from 0-4, 5-9, 42% percent mortality in 10-14 groups and 57.2% percent in 15-20 groups.

Scores for survivors was a mean of 7.72 and a standard deviation of 3.6, and for non survivors, mean of 16.14 and standard deviation of 2.96. $p = 5.79$, $p < 0.001$ which is statistically significant which compares with earlier studies by Adesunkanmi ARK, Badmus TA, Agbakwuru EA,[1,18] in adult African patients. Hence higher score indicates a need for concentration of medical services and expediting resources in treating those set of patients to reduce the morbidity and mortality.

Preoperative modified APACHE II scores are simple and effective method for assessing disease severity which is observed by our study.

CONCLUSION

Modified APACHE II scoring predicts mortality which was significant irrespective of the aetiology.

Higher Mean scores predicted serious morbidity outcomes.

Modified APACHE II Scores can be used easily and effectively to identify high risk patients for intensive therapy.

Modified APACHE II Scores can be used as a tool for surgical audit and research for improving the quality of intensive care in a hospital like ours.

BIBLIOGRAPHY

1. Adesunkanmi ARK, Badmus TA Agbakwuru EA, Acute Gen. Peritonitis in adult African patients, Assessment of severity using APACHE II. Ann. College of surgeon HK, 2003 : 7 : 23-8.

2. Bohnen J, Boulanger Meakins JL, Mclean PH Prognosis in Gen. Peritonitis, relation to cause and risk factors. Arch. Surg. 1983, 118 : 285-90.

3. Ponting GA, Sim AJW, Dudley, HAF, Comparision of local and systemic Sepsis in predicting survival, British J. Surg. 1987; 74 : 750-2.

4. Bion J, Outcome in Intestive care, BMJ, 1993, 307 ; 953-4.

5. Knaus WA, Drapper EA, Wagner DP, Zimmerman JE, APACHE severity of disease classification system. Crit. Care Med. 1985, 13 : 818-29.

6. Civetta JM, Hudson – Civetta SA, Nelson LD, Evaluation of APACHE II for cost containment and quality assurance. Ann. Surg. 1990, 212 : 266-76.

7. Baker SP, O'NeilB, Haddson W (u), Long WB, The injury severity score. A method for describing pattern of patients with multiple injuries and evaluation of emergency cases. J. Trauma, 1974; 14-187-96.

8. Greenspin L, Mclellan BA, Greig H, Abbrievated injury scale and injury severity score. A scoring chart J. Trauma, 1985; 25 : 60-4..

9. Copeland GP, Lones D, Walter M, Possum : A scoring system for surgical audit. Br. J. Surgery, 1991 : 78, 355-60.

10. Edwards AT. Ng. KJ. Shandall AA Prize Thomas JM Experience with APACHE II severity of disease scoring system in predicting outcome in surgical intensive therapy unit. Jr. Coll. Surg. Edinburgh, 1991 36 : 37-40.

11. Jones DR, Copeland GP, Decossart CL, Comparision of possum with APACHE II for prediction of outcome from a surgical high dependency unit. Br. J. Surg. 1992 : 79 : 1293-6.

12. Meakin JL, Solomkin JS, Allo MD, Dellinger PA, Proposed classification of intra abdominal infections. Stratification of etiology and risk of therapeutic trial. Arch. Surg. 1984 : 119-1372-8.

13. Cellan Jones C.J. A rapid method of treatment in perf. Duo. Ulcer. Br. Med. Journal, 1 : 1929.

14. Imhofen et al Journal of Chirugury, Paris 1987, 122 (6) 691-696.

15. Severity scores in surgery what for and who needs them. An introduction, definition, with classification and evaluation, Neugebaker EA, Legenberg, Arch Surg. 2002, Apr. 38 (1), 55-8.

16. Goffi L, Saba V, Ghiselli R, Newzione S, Pre op APACHE II and ASA in patients having major general surgery operations, prognostic value and potential clinical applications. European J. Surg. 1999, Aug. 165 (8) 730-5.

17. Demmel N Muth. G, Mang. K. Oster holzer G, Langerburgh Prognostic scores in peritonitis, the Mannhein peritonitis index or APACHE II, Arch. Surg. 1994 379 (6) 347 – 52.

18. Adesunkanmi Ak, Badmus TA, Fadiora FU, Agbakwuru EA, Generalised peritonitis secondary to typhoid ileal perforations assessment of severity using modified APACHE II scores, Indian. J. Surg. 2005, 67, 29-33.

19. Bailey and Love, Short practice of surgery, 24th Edition, B 67, P 1132-1134

20. Gray's Anatomy, 37th Edition, Peter L Williams, Mary Dyson, P 1342-47.

21. Autio V, The spread of intraperitoneal infection, Acta Chir, Scand. Suppl. 1981 : 95-98.

22. Maingot's Abdominal operations, Darry T. Hiyoma, Robert S. Bemison, 10th edition,

Vol. I, 637-638.

23. A.Cushieri RJ, Steel, R.R. Moosa, Essential surgical practice, IV Edition, Vol. I, (19.2) P. 445-446.

24. Results of diagnostic peritoneal tap in acute abdomen. Rao TN, Naik B, BB Indian Journal of Surgery, 1993, Jul. 55 (7), 338-341.

25. Clinical surgery by Somen Das, 33, P. 335-356.

26. Grasski R Ramano S Pinto A, Ramano L, Gastro duodenal perforation conventional plain film, USG, CT in 166 cases European journal of Radiology, 2004. Apr. 50(1) 30-6.

27. Mahanta H, Dasm K, Dutta Chowdary, An experience with diagnostic paracentesis in 100 cases of acute abdomen Journal of Indian med. Assoc. 1990, May 88(5), 125-9.

28. Boey J, Lee N, et al, Immediate definitive surgery for perforated duodenal ulcers. Annal. Of surgery 106; 338-1982.

29. Berne TV, Donovan AJ, Non operative treatment of perforated duodenal ulcer. Arch. Surgery 1989, 124(7); 830-2.

30. Crafts TJ, Dark KGM, Steel RK, et al, 1989, A randomized trial of non operative treatment for perforated peptic ulcer. New Eng.. J. Med. 320; 9780-973.

31. Lau WY, Lung KL Kwong KH et al 1996, A randomized study comparing laparascopic versus open repair of perforated peptic ulcer using suture or sutureless technique. Annal. of Surgery, 224 (131-138).

32. Donohue JH, Grass RA, Turnkey DD 1985, The management of duodenal and other small intestinal trauma. World. Journal of surgery, 9; 904-913.

33. General surgical operations, RM Kirk, Chapter 16, IV Edition, P321-323.

34. Thomas WEG, Willamson RCN, Enteric ulceration and its complications. World J. Surgery. 9 ; 8736, 1985.

35. Farqharson text book of operative surgery, 8th Edition, RF Rintoul, P. 455-456.

36. Hamilton Bailey's emergency surgery, 13th Edition, Chapter 27, P. 307-326.

37. Crawford E Ellis H, Generalised peritonitis – the changing spectrum. A report of 100 cases. Br. J. Clin. Practice 1985, 39 : 177.

38. Kachroo R et al Peritonitis (an analysis of 90 cases Indian journal of Surg. Vol. 46, April 1984.

39. Sharma L, Gupta S, Soin AS, Sikora S, Kapoor V, Generalised peritonitis in India – the tropical spectrum, Jpn J. Surg. 1991, May 21(3) 272-7.

40. Jones HJ, Decossart L, Risk scoring in surgical patients, Br. J. Surg. 1999, 86; 149-157.

41. Chong L, Han CF, Huang CC, Hsien YY, Prognostic accuracy of APACHE II SCORE in critically ill patients. American. J. Critical care, 2006, Jan 15(1) 47-53.

PROFORMA

PERFORATIVE PERITONITIS

ASSESSMENT OF SEVERITY USING

MODIFIED APACHE II SCORE

Name	Sex	Age
I.P. No.	Unit	Ward
Address	Occupation	
	DOA	DOD / Expired

PRESENTING ILLNESS

1. Abdominal Pain
    - Duration
    - Time of Onset
    - Mode of onset - Sudden / Gradual
    - Site of Pain
    - Shifting / Radiation / Referred pain
    - Character of Pain - Colicky / Constant
    - Relation of pain to movements
    - Any aggravating or relieving factor
2. Vomiting
3. Nausea
4. Abdominal distention
5. Fever
6. Anorexia
7. Diarrhea
8. Constipation
9. History of Trauma
10. Urinary Sympoms
11. Symptoms related to malignancy
12. Any other symptoms

PREVIOUS ILLNESS		Duration
1. Peptic ulcer	-	
2. Enteric Fever	-	
3. Appendicitis	-	
4. Dysentery	-	
5. Haematemesis	-	
6. Melaena	-	
7. Previous history of any surgery		
8. Recent Delivery / Abortion		
9. Any other illness in the past	-	HT / DM / TB

PERSONAL HISTORY

1. Appetite 2. Diet 3. Bowel Habit

4. Micturition habit 5. Smoking 6. Alcoholism

GENERAL EXAMINATION

1. Build & Nourishment
2. Level of consciousness
3. Attitude
4. Pulse rate
5. Blood Pressure
6. Respirator rate
7. Dysponoea
8. Pallor + or -ve
9. Icterus + or -ve
10. Cyanosis + or -ve
11. Temperature
12. Dehydration + or -ve

EXAMINATION OF ABDOMEN

Inspection

1. Abdominal distension
2. Movement with respiration
3. Visibl peristalsis
4. Henial Orifices
5. Position of Umbilicus
6. Flank Fullness- Present / Absent .
7. Injury - Present / Absent .

Palpation

1. Local rise of temperature
2. Tenderness
3. Abdominal guarding / Rigidity
4. Rebound tenderness
5. Liver
6. Spleen
7. Any other mass
8. Palpation of the hernial site

Percussion

1. Shifting Dullness
2. Fluid Thrill
3. Obliteration of liver dullness

Auscultion

Bowel sound - Present / Absent / Feeble / Increased

Per Rectal Examination

Per Vaginal Examination

Others systems

1. Cardio Vascular System
2. Respiratory System
3. Genitourinary System
4. CNS
5. Endocrine System

INVESTIGATIONS

A. Blood

Hb% Hematocrit
TC
DC
ESR
Blood - Urea / Glucose
Serum Creatinine
Widal
Blood Culture

B. Urine

Albumin
Sugar
Microscopy

Na+
K+
HCO_3

Serum Amylase
Blood Group

C. Chest X-ray

D. Plain X-ray Abdomen - Erect

E. Abdominal Paracentesis
- Aspirate Obtained /Not Obtained
- Morphological Appearance
- Peritoneal fluid culture & Sensitivity

MANAGEMENT
Conservative / Operative

OPERATION
Incision
Findings (a) Gas in peritoneal cavity - Present / Absent
(b) Nature of Fluid
(c) Orgains affected
(d) Other findings
Operative treatmnt given
Antibiotics used dose duration
Ryles tub aspiration - No. of Days
Bowel Sound - time of occurance
Post operative period - Uneventful/ Complications

COMPLICATIONS

RESULT
Cured / Expired

If expired cause of death

MODIFIED APACHE II SCORES

S.No.	Parameter	Value	Apache Points
1	Age		
2	Temperature		
3	Mean BP		
4	Heart Rate		
5	Respiratory Rate		
6	Na^+		
7	K^+		
8	Serum Creatinine		
9	Hematocrit%		
10	WBC		
11	HCO_3		

12	Serum Urea		
13	Chronic Health Points		
14	Emergency Surgery		